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=> file medline caplus biosis embase cancerlit

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FILE 'CANCERLIT' ENTERED AT 18:05:19 ON 22 DEC 2003

=> s (tnf or tumor (w) necrosis (w) factor) (w) antibodies L2 918 (TNF OR TUMOR (W) NECROSIS (W) FACTOR) (W) ANTIBODIES

=> 12 (S) (MIP-1b or RANTES) L2 IS NOT A RECOGNIZED COMMAND

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=> d 13 1- ibib, abs

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L3 ANSWER 1 OF 2 EMBASE COPYRIGHT 2003 ELSEVIER INC. ALL RIGHTS RESERVED.

on STN

ACCESSION NUMBER: 96342263 EMBASE

DOCUMENT NUMBER: 1996342263

TITLE: Tumor necrosis factor (TNF) in inflammatory bowel disease:

Gene polymorphisms, animal models, and potential for

anti-TNF therapy.

AUTHOR: Stokkers P.C.F.; Camoglio L.; Van Deventer S.J.H.

CORPORATE SOURCE: Lab. for Experimental Internal Med., Meibergdreef 9,1105 AZ

Amsterdam, Netherlands

SOURCE: Journal of Inflammation, (1995) 47/1-2 (97-103).

ISSN: 1078-7852 CODEN: JOINFB

COUNTRY: United States

DOCUMENT TYPE: Journal; Conference Article

FILE SEGMENT: 026 Immunology, Serology and Transplantation

LANGUAGE: English SUMMARY LANGUAGE: English

AB The inflammatory bowel diseases frequently require surgery because of intestinal complications. Animal models of inflammatory bowel disease, in particular those that histopathologically resemble Crohn's disease, are characterized by increased mucosal TNF production, and anti-TNF

antibodies have shown efficacy in decreasing disease activity. These data have provided a rationale for immunotherapy of Crohn's disease. Administration of anti-TNF antibodies to patients with Crohn's disease not responding to standard immunosuppressive treatment rapidly induced complete remissions, and healing of intestinal ulceration. A rapid reduction of circulating IL-6, CPR, and sPLA2 levels was observed in all patients, as well as a reduction of mucosal cells expressing RANTES and MIP-1. Short-term treatment with anti-TNF antibodies was not associated with significant toxicity, but results from long-term administration are still lacking. These data indicate that TNF is a pivotal and central inflammatory mediator in this disease. Further characterization of the precise mechanism of action of anti-TNF antibody therapy may further unravel the cause of immune dysregulaton in Crohn's disease.

ANSWER 2 OF 2 CANCERLIT on STN

ACCESSION NUMBER: 97071010 CANCERLIT

97071010 PubMed ID: 8913936 DOCUMENT NUMBER:

Tumor necrosis factor (TNF) in inflammatory bowel disease: TITLE:

gene polymorphisms, animal models, and potential for

anti-TNF therapy.

Stokkers P C; Camoglio L; van Deventer S J AUTHOR:

Laboratory for Experimental Internal Medicine, Academic CORPORATE SOURCE:

Medical Center, Amsterdam, The Netherlands.

JOURNAL OF INFLAMMATION, (1995-96) 47 (1-2) 97-103. Ref: SOURCE:

54

Journal code: 9511967. ISSN: 1078-7852.

PUB. COUNTRY: United States

Journal; Article; (JOURNAL ARTICLE) DOCUMENT TYPE:

General Review; (REVIEW)

(REVIEW, TUTORIAL)

English LANGUAGE:

MEDLINE; Priority Journals FILE SEGMENT:

MEDLINE 97071010 OTHER SOURCE:

199702 ENTRY MONTH:

Entered STN: 19970305 ENTRY DATE:

Last Updated on STN: 19970305

The inflammatory bowel diseases frequently require surgery because of AB intestinal complications. Animal models of inflammatory bowel disease, in particular those that histopathologically resemble Crohn's disease, are characterized by increased mucosal TNF production, and anti-TNF antibodies have shown efficacy in decreasing disease activity. These data have provided a rationale for immunotheraphy of Crohn's disease. Administration of anti-TNF antibodies to patients with Crohn's disease not responding to standard immunosuppressive treatment rapidly induced complete remissions, and healing of intestinal ulceration. A rapid reduction of circulating IL-6, CPR, and sPLA2 levels was observed in all patients, as well as a reduction of mucosal cells expressing RANTES and MIP-1. Short-term treatment with anti-TNF antibodies was not associated with significant toxicity, but results from long-term administration are still lacking. These data indicate that TNF is a pivotal and central inflammatory mediator in this disease. Further characterization of the precise mechanism of action of anti-TNF antibody therapy may further unravel the cause of immune dysregulation in Crohn's disease.

=> d his

(FILE 'HOME' ENTERED AT 18:04:54 ON 22 DEC 2003)

FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, CANCERLIT' ENTERED AT 18:05:19 ON 22 DEC 2003

L1333675 S TNF OR TUMOR (W) NECROSIS (W) FACTOR

918 S (TNF OR TUMOR (W) NECROSIS (W) FACTOR) (W) ANTIBODIES L2

CY

DТ

United States

Journal; General Review

=> s 11 S (fusion or conjugate) s chemokine? MISSING OPERATOR L1 S The search profile that was entered contains terms or nested terms that are not separated by a logical operator. => s s l1 S (fusion or conjugate) MISSING OPERATOR S L1 The search profile that was entered contains terms or nested terms that are not separated by a logical operator. => s l1 (s) (fusion? or conjugate?) 3699 L1 (S) (FUSION? OR CONJUGATE?) => s 14 (s) chemokine? 42 L4 (S) CHEMOKINE? => s 15 (s) antibodies 5 L5 (S) ANTIBODIES => duplicate remove 16 DUPLICATE PREFERENCE IS 'EMBASE, CANCERLIT' KEEP DUPLICATES FROM MORE THAN ONE FILE? Y/(N):n PROCESSING COMPLETED FOR L6 3 DUPLICATE REMOVE L6 (2 DUPLICATES REMOVED) L7=> d 17 1-YOU HAVE REQUESTED DATA FROM 3 ANSWERS - CONTINUE? Y/(N):y ANSWER 1 OF 3 EMBASE COPYRIGHT 2003 ELSEVIER INC. ALL RIGHTS RESERVED. L7on STN 2003161017 EMBASE AN Cytokine, chemokine, and costimulatory molecule modulation to enhance TIefficacy of HIV vaccines. Ahlers J.D.; Belyakov I.M.; Berzofsky J.A. ΑU J.A. Berzofsky, Molec. Immunogen./Vaccine Res. Sec., Metabolism Branch, CS National Cancer Institute, Building 10, Bethesda, MD 20892-1578, United States. jahlers@niaid.nih.gov Current Molecular Medicine, (2003) 3/3 (285-301). SO Refs: 206 ISSN: 1566-5240 CODEN: CMMUBP CY Netherlands DTJournal; General Review Microbiology FS 004 Immunology, Serology and Transplantation 026 Pharmacology 030 037 Drug Literature Index 039 Pharmacy LA English SLEnglish ANSWER 2 OF 3 EMBASE COPYRIGHT 2003 ELSEVIER INC. ALL RIGHTS RESERVED. L7 DUPLICATE 1 on STN 2001154626 EMBASE ΑN Anti-TNF.alpha. therapy of rheumatoid arthritis: What have we learned?. TIAU Feldmann M.; Maini R.N. CS M. Feldmann, Kennedy Inst. of Rheumatology Div., Imperial College School of Medicine, 1 Aspenlea Road, London W6 8LH, United Kingdom. m.feldmann@ic.ac.uk Annual Review of Immunology, (2001) 19/- (163-196). SO Refs: 160 ISSN: 0732-0582 CODEN: ARIMDU

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Immunology, Serology and Transplantation
FS
     026
             Arthritis and Rheumatism
     031
     037
             Drug Literature Index
     English
LΑ
SL
    English
     ANSWER 3 OF 3 EMBASE COPYRIGHT 2003 ELSEVIER INC. ALL RIGHTS RESERVED.
L7
                                                         DUPLICATE 2
     on STN
     1998362492 EMBASE
AN
     Fractalkine and CX3CR1 mediate a novel mechanism of leukocyte capture,
TI
     firm adhesion, and activation under physiologic flow.
     Fong A.M.; Robinson L.A.; Steeber D.A.; Tedder T.F.; Yoshie O.; Imai T.;
ΑU
     D.D. Patel, Box 3258, 222 CARL Bldg., Duke University Medical Center,
CS
     Durham, NC 27710, United States. patel003@mc.duke.edu
     Journal of Experimental Medicine, (19 Oct 1998) 188/8 (1413-1419).
SO
     ISSN: 0022-1007 CODEN: JEMEAV
     United States
CY
     Journal; Article
DT
             Hematology
     025
FS
             Clinical Biochemistry
     029
     English
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m SL}
=> d his
     (FILE 'HOME' ENTERED AT 18:04:54 ON 22 DEC 2003)
     FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, CANCERLIT' ENTERED AT 18:05:19 ON
     22 DEC 2003
         333675 S TNF OR TUMOR (W) NECROSIS (W) FACTOR
L1
            918 S (TNF OR TUMOR (W) NECROSIS (W) FACTOR) (W) ANTIBODIES
L2
              2 S L2 (S) (MIP-1B OR RANTES)
L3
           3699 S L1 (S) (FUSION? OR CONJUGATE?)
L4
L5
             42 S L4 (S) CHEMOKINE?
              5 S L5 (S) ANTIBODIES
L6
              3 DUPLICATE REMOVE L6 (2 DUPLICATES REMOVED)
L7
=> log y
                                                                  TOTAL
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FULL ESTIMATED COST
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